

Amendments to the Specification:

Please amend the specification as follows:

Please append the enclosed Sequence Listing filed concurrently herewith in the Specification after page 15.

Please replace the following paragraph beginning on page 2, line 20:

A pharmaceutical composition containing a peptide or pharmaceutically acceptable salt thereof containing the amino acid sequence LLGDFFRKSKEKIGKEFKRIVQRIKDFLRNLVPRTES (LL-37) (SEQ ID NO: 1) and a carrier is disclosed. A method of increasing angiogenesis is also disclosed and involves administering a pharmaceutical composition containing a peptide or pharmaceutically acceptable salt thereof containing the amino acid sequence LLGDFFRKSKEKIGKEFKRIVQRIKDFLRNLVPRTES (LL-37) (SEQ ID NO: 1).

Please replace the following paragraph beginning on page 3, line 18:

Methods and pharmaceutical compositions are disclosed containing a peptide having the amino acid sequence LLGDFFRKSKEKIGKEFKRIVQRIKDFLRNLVPRTES (also termed LL-37) (SEQ ID NO: 1) for the preparation of a pharmaceutical composition for the prevention or treatment of a disease caused by or resulting in a reduced level of angiogenesis or of arteriogenesis, or for the treatment of wounds.

Please replace the following paragraph beginning on page 4, line 24, through page 5, line 2:

Nucleic acid molecules specifically hybridizing to LL-37 or its receptor encoding genes and/or their regulatory sequences may be used for repression of expression of said gene by methods as disclosed in e.g. PCT/DE00/00244, PCT/US98/27233 or PCT/US01/10188, or for example due to an antisense or triple helix effect or they may be used for the construction of appropriate ribozymes (see, e.g., EP-B1 0 291 533, EP-A1 0 321 201, EP-A2 0 360 257) which specifically cleave the (pre)-mRNA of a gene encoding LL-37. The nucleic acid sequences encoding LL-37 are known in the art and described, for example, in GeneBank, accession number: Protein P49913 (SEQ ID NO: 2), mRNA NM_004345 (SEQ ID NO: 3), gene X96735 (SEQ ID NO: 4). Selection of appropriate target sites and corresponding ribozymes can

be done as described for example in Steinecke, Ribozymes, Methods in Cell Biology 50, Galbraith *et al.* eds Academic Press, Inc. (1995), 449-460.

Please replace the following paragraph beginning on page 12, line 14:

Total RNA was isolated from endothelial cells from human umbilical veins HUVECs (Trizol reagent, LifeTechnologies, Karlsruhe, Germany) and reverse transcribed (Superscript II reverse transcriptase system, LifeTechnologies). Primers were designed to amplify FPRL1 cDNA based on the GenBank file (accession number NM001462): sense: 5-GAC CTT GGA TTC TTG CTC TAG TC-3' (SEQ ID NO: 5); antisense: 5'-CCA TCC TCA CAA TGC CTG TAA C-3' (SEQ ID NO: 6). Primers for glyceraldehyde-phosphate-dehydrogenase (GAPDH) were used as positive control. PCR conditions were applied as described earlier⁷ using a RoboCycler Gradient 40 with hot top (Stratagene, Heidelberg, Germany). Products were separated by agarose gel electrophoresis, stained with ethidium bromide and viewed on a UV transilluminator.

Please replace the following paragraph beginning on page 12, line 26, through page 13, line 11:

Based on these results we investigated whether agonists of FPRL1 induce reactions of endothelial cells. Endothelial cells from human umbilical veins (HUVEC) were isolated as described before⁸. 10⁴ cells were seeded on six well plastic dishes in serum free medium. Different concentrations of LL-37 were added to the culture medium. VEGF (5 ng / ml, 165 amino acid variant, Sigma Chemicals, Munich, Germany) was used as positive control. After 72 h the cells were detached from the support and counted. Experiments were repeated ten times using synthetic LL-37/hCAP-18 (37 C-terminal amino acids) and the hexapeptide WKYMVm (SEQ ID NO: 7), wherein "m" denotes the D-form of the amino acid methionine, from two different sources (IPF Pharmaceuticals, Hannover, Germany and Institute for Biochemistry, Humboldt-University of Berlin, Germany). As control peptide we used the peptide L-14 unknown to have any interactions with FPRL1 from the same source (Humboldt-University). Human serum (10 %, donor with ABO blood group O) was applied under the same conditions to analyze any impact on induction of proliferation by LL-37. For inhibition experiments we applied inhibitors of G-protein coupled receptors (pertussis toxin, 10 ng/ml), PI-3K (wortmannin), and PKC (GF 109203X). For Matrigel assays HUVECs were seeded on Matrigel

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support (Beckton Dickinson, Franklin Lakes, NJ) and cultivated in serum free medium in the absence or presence of 0.5 µg/ml LL-37. After 18 h, the cultures were inspected for the formation of rings and cords. Experiments were repeated three times.

Please replace the Abstract on page 19:

A pharmaceutical composition containing a peptide or pharmaceutically acceptable salt thereof containing the amino acid sequence LLGDFFRKSKIGKEFKRIVQRIKDFLRNLVPRTES (LL-37) (SEQ ID NO: 1); for preventing or treating wounds, tumors, or disease caused by or resulting in a reduced level of angiogenesis or of arteriogenesis is disclosed.